

Intra-articular Disc Displacement Part I: Its Questionable Role in Temporomandibular Joint Pathology

Disc Displacement
CAUSES PAIN

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The publications by Farrar relating temporomandibular joint (TMJ) signs and symptoms to disc displacement, and the rediscovery of TMJ arthrography by Wilkes, has stimulated renewed interest in TMJ internal derangement.^{1,2} During the 1970s and 1980s, numerous publications documenting disc displacement and its clinical presentation appeared in the literature.³⁻⁷ Supporting clinical evidence for disc displacement came from diagnostic imaging and surgical observations correlated with clinical signs and symptoms. It was proposed that TMJ pain, mandibular dysfunction, osteoarthritis, and mandibular growth disturbances could be attributed to displacement of the TMJ disc and the position and shape of the disc became the focus of classification, diagnosis, and treatment of TMJ pain and dysfunction. However, despite the clinical evidence supporting the existence of TMJ disc displacement, there remained many unanswered questions that raised doubt as to the significance of such displacement.

In 1983, I coauthored an article, "Internal Derangement of the Temporomandibular Joint: Fact or Fiction" in which it was concluded that TMJ disc displacement was a reality and, further, the major cause of TMJ pain, dysfunction, and osteoarthritis.⁸ However, during the past decade much has been learned about TMJ internal derangement. The purpose of this article is to re-evaluate the relationship of disc position to pain, mandibular dysfunction, osteoarthritis, and growth disturbances, ie, to reconsider the clinical significance of disc displacement.

Relationship of Disc Displacement to Pain

The most perplexing question is, "What is the relationship of disc displacement to pain?" Because pain

in patients with disc displacement is usually aggravated during movement, it seems reasonable that the pain originates from pressure and traction on the disc attachments. In joints with anterior disc displacement, the loose, vascular, innervated tissues in the posterior attachment are displaced between the condyle and fossa and could be compressed or stretched during function. However, there is still the question of why many, perhaps most, patients with disc displacement have no pain while some have severe pain.

TMJ clicking or popping has been shown to occur in 30% to 50% of the population. Most individuals with clicking joints probably have some form of disc displacement, yet most of these people do not have pain.⁹ The presence of clicking is generally found to be evenly distributed between men and women, yet pain is reported much more often by females. It has also been shown that when normal TMJs, ie, free of signs and symptoms, are studied arthrographically or with magnetic resonance imaging (MRI), approximately 30% of the joints show evidence of disc displacement.^{10,11} It has also been observed that when bilateral arthrograms are performed on patients with unilateral symptoms, the nonpainful joint demonstrates evidence of disc derangement 88% of the time.¹² The most compelling evidence against the displaced disc being the cause of TMJ pain comes from the observations that arthroscopy and arthrocentesis of the superior joint compartment reduces or eliminates pain in patients with closed-lock without repositioning the displaced disc.¹³⁻¹⁵ These findings make it obvious that while disc displacement may exist, it is not necessarily related to pain. *

Relationship of Disc Displacement to Dysfunction

The relationship of disc displacement to reciprocal clicking, when the click occurs at different positions during the opening and closing movements, is clear, and the evidence supporting this relationship is strong.

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Using arthrography, it has been shown that as mouth opening occurs, the disc is pushed anteriorly until a click occurs, at which time the disc returns to a normal relationship with the condyle.^{2,4} It was also observed that during the closing movement, the disc is again displaced anteriorly. Further, the reduction and displacement of the disc correlated with the clinical finding of reciprocal clicking. The anatomic events associated with reciprocal clicking were confirmed by Isberg-Holm using high speed cinematography on autopsy specimens.⁷

TMJ arthrography performed on patients with closed-lock demonstrated that the disc was displaced anteriorly and remained displaced throughout the opening and closing cycle.^{2,4} This led to the obvious conclusion that the displaced disc was the cause of the observed limited opening. However, the observation that arthroscopy or arthrocentesis involving lavage of the superior joint compartment without repositioning the disc re-established normal mouth opening in patients with closed-lock has seriously questioned this conclusion.¹³⁻¹⁵ If normal range of motion can be re-established without repositioning the displaced disc, then alternative explanations for closed-lock are plausible.¹⁶

Relationship of Disc Displacement to Osteoarthritis

The relationship of disc displacement to osteoarthritis is controversial. Studies of human TMJs have provided evidence that disc displacement is associated with an increased incidence of osteoarthritis.⁶ Osteoarthritis of the TMJ has also been documented in more than 50% of patients with disc derangement. With imaging studies, osteoarthritis is rarely observed in joints with normal disc position, but is often observed in joints with disc perforation and severe deformation.¹⁷ These results support the idea that disc displacement precedes osteoarthritis and that disc displacement may be a cause of osteoarthritis. On the other hand, early osteoarthrotic changes such as articular cartilage softening and fibrillation have been observed arthroscopically in the TMJs with normal disc position or minimally displaced discs. Additionally, DeBont observed histologically "osteoarthrotic changes" affecting the articular surfaces of the TMJ in 4 of 8 joints with normal disc-condyle relationships.¹⁸ These results support the idea that osteoarthrotic changes precede disc displacement and that disc displacement may be a sign of osteoarthritis and not its cause.

Although it appears that disc displacement and osteoarthritis occur together, it is still unclear whether disc displacement precedes or follows osteoarthritis. It is possible that both pathways exist. A displaced

disc may cause the development of osteoarthritis or osteoarthritis may cause disc displacement. It is also possible that they occur together but are unrelated.

Relationship of Disc Displacement to Mandibular Growth Disturbance

It has recently been stated that disc displacement is a significant cause of mandibular growth disturbance.¹⁹ Imaging studies have shown disc derangement in association with mandibular deficiency and asymmetry.²⁰ These studies were reported to support earlier observations by Boering and Ricketts.^{21,22} Conversely, not all growing patients who have disc displacement grow abnormally, nor do all patients with growth deficiencies have disc displacement. It would seem that if disc displacement were a significant cause of mandibular growth deficiency, the signs and symptoms of disc displacement would be more common in this population than in the normal population. The evidence does not support this and, in fact, it has been observed that the signs and symptoms of disc displacement do not occur more commonly in dentofacial deformity patients.²³ Thus, whether or not disc displacement is a cause of growth disturbance needs to be investigated further.

Discussion

The relationship of disc displacement to pain, mandibular dysfunction, osteoarthritis, and growth disturbance remains unclear. Alternative explanation for these problems are plausible and should be considered. Observations during TMJ arthroscopy and outcomes after such treatment have provided new insights into the pathology of TMJ internal derangement. For example, the role of inflammation in the TMJ was not appreciated until the use of TMJ arthroscopy. Signs of inflammation, eg, hypervascularity and erythema, are commonly observed and have been shown to correlate with the severity of the pain.²⁴ Other evidence of inflammation, eg, joint effusion, has been demonstrated with T2-weighted MRIs,²⁵ and such effusion has also been shown to be related to pain. While disc displacement may be a cause of pain, inflammation is clearly another cause.

Disc displacement correlates well with reciprocating clicking, but alternative explanations exist for closed-lock. Fibrous adhesions observed during TMJ arthroscopy have been shown to be an important cause of limited opening.²⁶ Release of fibrous adhesions has restored normal range of motion in many patients with closed-lock even when the disc has not been repositioned. Obviously, fibrous adhesions are an alternative cause of closed-lock. Other proposed causes of closed-lock include the suction-cup effect, the negative pres-

sure (vacuum) effect, and increased friction and low viscosity of the synovial fluid.^{13,15-16}

A relationship between disc displacement and osteoarthritis has been shown by several investigators. However, controversy still exists as to whether disc displacement is the cause or result of osteoarthritis. Articular cartilage and the underlying bone display a dynamic equilibrium between changes in form and function involved in tissue remodeling. The tissues will adapt to applied stress but, when the stress or loading is excessive, tissue breakdown may occur. Severe overloading can cause irreversible changes and damage to the articular cartilage with the release of degradation products into the synovial fluid, possibly causing synovitis. Additionally, many proteases, cytokines, growth factors, and arachidonic acid metabolites play a role in the pathogenesis of osteoarthritis.²⁷ The causes of osteoarthritis, although not clearly understood, are complex and multifactorial, and cannot be explained simply by disc displacement.

The evidence that disc displacement may cause a mandibular growth disturbance generally comes from small groups of TMJ patients with concomitant growth disturbances who were evaluated with various TMJ imaging techniques. Control groups were not included for comparison, nor were alternative explanations evaluated. The evidence that TMJ problems are no more common within the dentofacial deformity population than the general population raises doubt as to disc derangement being a major cause of mandibular growth disturbances. As with osteoarthritis, growth disturbances probably occur as a result of many factors including genetic, traumatic, pathologic, and possibly disc derangement.

During the past 20 years much has been learned about TMJ internal derangement. Evaluation of this information indicates that the role of disc displacement as the primary pathologic factor may not be justified. Although much research remains to be done, the evidence strongly suggests that TMJ internal derangement is a much more complicated process than simply a disc displacement; TMJ internal derangement also involves inflammation, changes in the articular surfaces, alteration in joint pressures and synovial fluid, production of a variety of biochemical substances, and possibly several yet to be defined factors. Consequently, the focus of classification, diagnosis, and treatment of TMJ internal derangement on disc position should be re-evaluated. As stated by Thomas Hunt Morgan in the book *Experimental Embryology*, "The investigator must . . . cultivate also a skeptical state of mind toward all hypotheses—especially his own—and be ready to abandon them the moment the evidence points the other way."

Treatment of TMJ internal derangement generally has been directed at procedures, both nonsurgical and

surgical, designed to reposition the displaced disc. Obviously, a clinician's beliefs about the importance of disc displacement determines his or her philosophy of treatment. In view of the new evidence about TMJ internal derangement, treatment should be directed at pain management, reduction of inflammation, decreasing adverse joint loading, and restoration of normal range of motion rather than repositioning the disc.

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